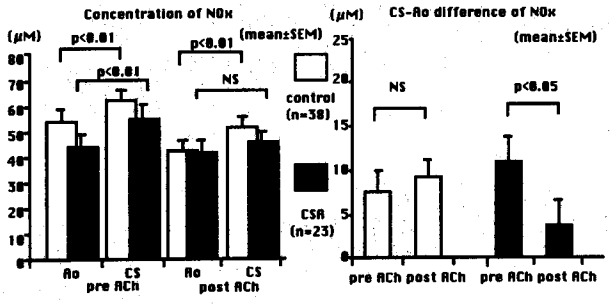
was higher in CS than Ao before ACh provocation. In the control group, the concentration of NOx was also high in CS after ACh provocation. However, in CSA group, NOx was not high in CS. The CS-Ao difference of NOx was increased after ACh provocation in the control group. On the other hand, it was decreased in CSA patients.



The results are shown in figures. These results suggests that there is impairment of NO product in CSA patients.

1096-81 Serum Lipids and Hypertension Potentiate Coronary Vasoconstriction with Mental Stress

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In angiographically normal coronary segments (NORM) paradoxical coronary constriction to acetycholine occurs in the presence of elevated LDL and serum cholesterol. To determine the effect of serum lipids and other risk factors, on coronary vasomotor response to mental stress (MS), we performed quantitative coronary angiography in 34 male CAD pts (av age 60 ± 10 yrs) before and immediately after mental arithmetic with harrassment. With MS, there were increases (all $p < 0.0001$) in mean BP (13 ± 10 mmHg) and HR (13 ± 10 bpm). In NORM segments coronary diameter (DIAM) change with MS ranged from −13.2% (constriction) to +12.4% (dilation). Change in DIAM was inversely correlated with total cholesterol ($r = -0.52$, $p < 0.002$) and LDL ($r = -0.48$, $p < 0.004$). In pts with LDL > 190 mg/dl (av 230 ± 19), DIAM decreased 5.6 ± 4.5%, in contrast to increase ($p = 0.0001$) of DIAM 2.1 ± 4.3% in pts with LDL ≤ 190 mg/dl (av 135 ± 26). There was a 4.6 ± 4.8% difference in vasomotion with MS between pts with and without hypertension ($p = 0.01$); absence of hypertension was associated with dilation (3.3 ± 5%, $p = 0.03$). Coronary vasomotor response to MS was not related to age, smoking status, # of diseased vessels, or family history of CAD. In atherosclerotic segments, however, neither lipoproteins nor CAD risk factors were associated with coronary vasoconstriction. *Conclusion:* Serum lipids and hypertension promote coronary vasoconstriction with MS in angiographically normal, but not overtly atherosclerotic segments.

1096-82 Impaired Functional Capacity in Coronary Microcirculation in Patients With Diabetes Mellitus

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Structural abnormality in coronary microvasculature has been reported to be present in animal models and pts with diabetes mellitus (DM) even in the absence of significant stenosis in epicardial coronary arteries. To clarify whether such structural abnormality is associated with functional abnormality in coronary microcirculation, coronary blood flow velocity was measured with aortic blood pressure using a 0.014″ Doppler guide wire in 11 DM pts and 8 pts without DM before and after intracoronary infusion of ATP. Then, the instantaneous coronary blood flow velocity was plotted against the simultaneously measured aortic pressure, and the slope of the velocity-pressure relation in the phase of progressive diastolic velocity decrease (F/P, cm/s/mmHg) was calculated. F/P increases with a decrease in coronary resistance and has been reported to be less affected by loading conditions than coronary flow reserve. None of these 19 pts had significant stenosis of epicardial coronary arteries. Results were following:

	C-F/P	A-F/P	F/P ratio
pts without DM	0.6 ± 0.1	2.3 ± 0.5*	3.0 ± 0.9
DM pts	0.8 ± 0.2	1.4 ± 0.4*	1.1 ± 0.3#

(mean ± SEM, C-F/P = F/P before ATP infusion, A-F/P = F/P after ATP infusion, F/P ratio = a ratio of A-F/P to C-F/P, *$p < 0.05$ vs C-F/P, # $p < 0.05$ vs without DM)

In both pts, F/P significantly increased with ATP Infusion. However, in DM pts A-F/P tended to be lower and F/P ratio was significantly lower than in pts without DM. *Conclusion:* Functional capacity in coronary microcirculation is impaired in pts with DM even in the absence of significant stenosis of epicardial coronary arteries.

1096-100 The Effect of Hypercholesterolemia on Intimal Thickness and Endothelial Function after Heart Transplantation

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The non-immunological risk factor cholesterol may aggravate structural or functional manifestations of epicardial and/or microvascular transplant vasculopathy (TXV). We investigated 27 patients 47 ± 10 month after HTX using Intracoronary Ultrasound (ICUS; 2.9 F; 30 MHz; measurement of mean intimal thickness (MIT) and intimal index) and Doppler (Flowire, 12 MHz). Relative coronary flow reserve (CFR) during intracoronary acetylcholine infusion (ACM, 30 μg/min over 5 min) reflected microvascular endothelial function (MEF). Epicardial vasoconstriction (EV) during ACH (quantitativ angiography) was defined as abnormal epicardial endothelial function (EEF). For each patient the mean of 12–15 at regular intervals determined serum cholesterol levels (Chol, LDL-C, HDL-C) were used to represent postoperative lipid profile.

Results:

	n	mg/dl	MIT (μm)	CFR (%)	EV (% of pts)
LDL-C > 110 mg/dl	21	142 ± 17	370 ± 205	228 ± 82	24
LDL-C ≤ 110 mg/dl	6	103 ± 7***	170 ± 184*	189 ± 22	25
HDL-C ≥ 55 mg/dl	8	71 ± 16	172 ± 178	227 ± 50	0
HDL-C < 55 mg/dl	19	45 ± 6***	390 ± 199**	217 ± 88	36*

*$p < 0.05$; **$p < 0.02$; ***$p < 0.0001$

HDL-C but not CHOL, LDL-C or LDL-C/HDL-C correlated weakly with MIT ($r = -0.45$, $p < 0.02$) and intimal index ($r = -0.47$, $p < 0.02$).

Conclusions: Low LDL-C and high HDL-C are associated with significant less intimal thickness in the long term course after HTX. MEF is unaffected by LDL-C or HDL-C; EEF seems to be protected by high HDL-C. An aggressive lipid-modulating therapy should be performed to ameliorate epicardial morphology and function in TXV.

1096-101 Heterogeneous Vasomotor Responses of Conduit and Resistance Coronary Arteries after Intracoronary Acetylcholine in Hypertension

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Hypertension (HTN) is associated with changes in vasoreactivity of conduit (CON) and resistance (RES) coronary arteries in response to the endothelium dependent agent acetylcholine (ACh). The purpose of this investigation was to determine the correlation between vasoreactivity of CON and RES coronary arteries among normotensive (NTS) and hypertensive (HTS) subjects. Subjects underwent endothelial function and coronary reserve testing after referral for cardiac cath for evaluation of chest pain. Percent increase in coronary blood flow (CBF) after ACh was assumed to primarily represent relaxation of the RES arteries. This was plotted vs % increase in CON artery diameter after ACh. In 31 NTS, nondiabetic patients, RES and CON artery vasoreactivity after ACh were significantly correlated ($r = 0.69$, $p = 0.00002$), though 7 pts (23%) had constriction of CON but dilatation of RES arteries. In 28 HTS pts without LVH, RES and CON artery responses were correlated ($r = 0.44$, $p = 0.01$), though 11 pts. (39%) had constriction of CON but dilatation of RES arteries. In 39 HTS pts with LVH, responses were again correlated ($r = 0.46$, $p = 0.003$), though 19 pts (49%) had discordant findings. NTS and HTS subjects without LVH had similar peak increases in CBF after ACh (212 ± 22% and 193 ± 28%) but HTS subjects with LVH had marked depression in this response (95 ± 11%).

Conclusions: Despite nl CON arteries, heterogeneous responses of CON and RES coronary arteries to ACh are found in pts referred for cardiac cath because of chest pain. Greater frequency of discordant CON and RES artery responses are found with increasing severity of HTN. This data suggests that HTN affects CON vessels earlier and more prominently than RES vessels. With the advent of LVH, however, both RES and CON arteries exhibit impairment in ACh induced vasodilation.